Thrombosis/Thromboembolism of the descending aorta (ATE) in dogs: diagnosis, pathophysiologic and therapeutic considerations

Glaus, T M

Thrombosis / Thromboembolism of the descending aorta (ATE) in dogs, diagnosis, pathophysiologic and therapeutic considerations.

Tony Glaus, Prof. Dr. med. Vet., Dipl. ACVIM and ECVIM-CA, Head Division of Cardiology, Vetsuisse Faculty University of Zurich, CH-8057 Zurich

Definitions

Thrombosis is the local formation of a thrombus inside a vessel leading to vascular occlusion, embolism is the vascular occlusion by various formations like a thrombus, tissue particle, parasite, foreign body, tumor cells or air that have been formed distant to the actual occlusion and that are carried in the vessel to the site of occlusion, and thromboembolism is thus the vascular occlusion specifically by a thrombus that was not produced at the site of occlusion but carried there. Once a thrombus is present in the circulation it may grow. In this text, the abbreviation ATE will be used to describe both aortic thrombosis and / or thromboembolism.

Pathogenesis

For ATE to occur an important disturbance in the delicate balance between pro- and antithrombotic body systems has to be present, i.e. there may be excessive abnormal focal vascular triggers of coagulation, or a systemic hypercoagulable state, or a systemic hypofibrinolytic state, or a combination of these. The following notes focus on ATE in dogs. As opposed to cats, ATE is not a very common problem in dogs. Furthermore, causes of ATE markedly differ in cats and dogs. In cats, around 90% of cases are due to thrombus formation in the left atrium (or ventricle) secondary to stagnant blood flow, mostly due to a severely dilated left atrium secondary to myocardial disease. Around 10% are due to cancer, most commonly carcinomata in the chest. In dogs, ATE is not encountered with atrial dilation, even if severe. As a matter of fact, in many cases no eliciting cause is identified. In some dogs ATE has been reported associated with local vascular pathology, specifically atherosclerosis in the descending aorta, or (adrenal) cancer breaking into the aorta. Furthermore, it has been associated (published and unpublished) with systemic diseases, including Leishmaniosis, nephrotic syndrome (some due to leishmaniasis), protein-losing enteropathy, hypothyroidism, hyperadrenocorticism, endocarditis, ectopic heart worms, cancer, unspecified nephropathy and unspecified cardiac disease.

Clinical signs, differential diagnosis and further diagnostics

Whereas in some dogs, clinical signs may be peracute with pain, paraplegia, cold extremities, paresthesia and no palpable pulse, like in cats, more commonly the onset of gait abnormalities may be insidious and worsen progressively. A femoral pulse may still be palpable delaying the suspicion of ischemic myopathy as cause of the gait abnormality. Characteristic for narrowing of the aorta and decreased oxygen delivery to the rear legs is exercise induced intermittent claudication. The differential diagnosis for this includes right-to-left PDA, myasthenia gravis, neuropathies and myopathies. When a pulse is still palpable, it takes a high index of suspicion to consider ATE. In some cases, ATE is found incidentally during abdominal ultrasound. The minimal data base to search for a potential underlying cause includes routine hematology, biochemical profile, urinalysis, urine-proteine-creatinine-ratio, thoracic radiographs and abdominal ultrasound. The latter also is necessary to prove the diagnosis. Further tests may include infectious disease titers (Leishmania), and abdominal CT.

Treatment

The treatment aims at 1. symptomatically fight the pain, 2. eliminate the thrombus, 3. eliminate the underlying cause.

To eliminate the thrombus medically, fibrinolytic and antithrombotic therapy are possible. Complications with both include

- breaking off of part of the thrombus, which may cause complete arterial obstruction lower in the arteries, which may lead to paraplegia of a previously ambulatory patient.
- Spontaneous bleeding.

In a patient with acute paraplegia and pulselessness of very recent onset, we would consider fibrinolytic treatment using tissue plasminogen activator. In this scenario, an additional risk is severe hyperkalemia secondary to acute reperfusion. In a still ambulatory patient, our approach today is the application of the low-molecular-weight heparin enoxaparine (0.8 mg/kgq6h, Clexane^{R}) combined with the antiplatelet drug clopidogrel (2-3 mg/kg q24h, Plavix^{R}). To assure that the enoxaparine blood level is in the target range, anti-factorXa-activity is assessed using the human assay. When the thrombus is completely dissolved based on ultrasonographic examination of the descending aorta, the enoxaparine dose is slowly decreased, but never completely stopped. Enoxaparine is expensive.

Prognosis

Basically poor, but better than in cats. Depends on underlying cause, onset acute versus chronic, still ambulatory versus paraplegic, and severity of secondary complications like

neuropathies, muscle necrosis / fibrosis. Complete recanalisation is possible, however, breaking off of parts of the thrombus may occlude distal arteries with acute paraplegia in a previously walking dog. Relapses are possible.

References

Boswood A et al. Aortic and iliac thrombosis in six dogs. J Small Anim Pract 2000;41:109-114.

Goncalves R et al. Clinical and neurological characteristics of aortic thromboembolism in dogs. J Small Animl Pract 2008; 49:178-184.

Lunsford KV et al. Pharmacokinetics of Subcutaneous Low Molecular Weight Heparin (Enoxaparin) in Dogs. J Am Anim Hosp Assoc 2009; 45: 261-267.